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ACUTE PERFORATED GASTRIC AND DUODENAL ULCERS*

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Perforation of gastric and duodenal ulcer may be acute, sub-acute, or chronic. In this paper we will deal only with the acute, so when perforated is used herein it shall designate acute perforation.

Perforation occurs in about 7 to 15% of the chronic gastric or duodenal ulcers. Sippy [1] believes perforation occurs in about 7% of the ulcer cases. Deaver [2] says, "Acute perforating, peptic ulcers occurs in 15 to 20% of ulcer patients."

In perforated ulcer we are dealing with an acute condition ingrafted upon a chronic ulcer, that is, the ulcer is practically always chronic, the perforation always acute. Moynihan [3] has seen only one acute ulcer in fifty cases of perforation. We have in perforated ulcer, a disease in which the male sex has the monoply, one series showing fifty-four males and one female, while in our series all were males. In Deaver's [4] series seventeen were gastric and thirty-eight duodenal ulcers. In our series four were gastric and twelve were duodenal ulcers. The average age is about forty years. The average in our series is forty-three years, the youngest patient being twenty-one and the oldest fifty-eight years.

Harrigan [5] believes that there is a distinctive type of ulcer that has a tendency to perforation. Wilensky [6] holds that embolism occurs in the crater of a chronic ulcer with resulting shutting off of the blood supply allowing digestive action of gastric juice to cause an acute perforation.

The majority of cases give a history of previous stomach trouble, either a typical gastric or duodenal ulcer history or a story of a long standing, obstinate "dyspepsia." Often there has been treatment for ulcer. Occasionally, there is no history of previous gastric distress. Cases have perforated while in the hospital under observation.

In a typical case of perforating ulcer, there are warning symptoms several days prior to the perforation. These consist of an exaggeration of the previous symptoms. The onset is often ushered in following a heavy meal, trauma or straining. In one cases of our series, the attack occurred as the patient was taking an enema.

The first symptom in time and importance is pain in the epigastrium. It is the pain of a ruptured viscus, which is severe, agonizing and almost unbearable. There is no other pain that is as excruciating. The patient is pale and prostrated, his face is anxious and there is a frightened expression in his eyes. Beads of sweat stand out on the brow. The breathing is short and rapid, deep inspirations causing severe pain. He is motionless, holding his entire body rigid, resenting being moved. His requests for relief are urgent and he is loathe to be examined, as the least pressure is painful. The abdomen is normal contour. The muscular rigidity is of the highest degree—board-like, never relaxing, persisting thus until near death in the fatal cases. It is unaffected by posture or pressure. In the gastric cases an epigastric area of special tenderness may be found. This is not true in the perforated duodenal ulcer cases, the tenderness in the latter being especially in the epigastrium and lower right quadrant. Vomiting is often present but is not constant. Diminished or absent liver dullness is found many times.

From the appearance of the patient one would expect to find him in marked shock. Such is not the case. The patients seen early are in especially good condition. The pulse is rarely above 90. The temperature is normal or sub-normal. The blood pressure is practically unchanged. The leukocyte count is raised only slightly, the average W.B.C. being 13,000. The respirations are rapid and shallow. This condition of pulse, blood pressure and temperature

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does not continue long before a change is noticed. We should not be confused and make the error of delay by waiting to see if there will be a raise in temperature or an acceleration of the pulse, as it is during the early stage that surgery gives the best results.

The mainstays of the diagnosis, then, are a well taken history, and 2 abdominal signs—that is, board-like and parallel tenderness. There are few other conditions in which the history is more important.

Lohr [7] observing one hundred thirty-two cases has concluded that the first twelve hours the peritonitis is chemical; the acid gastric juice prevents growth of intestinal bacteria. After this period, alkalescence prevails, allowing pathogenic intestinal bacteria to flourish producing bacterial peritonitis.

Deaver obtained twenty-three sterile cultures out of thirty-four cases; Brenner [8] obtained eleven negative cultures out of fifteen cases. The concurrence of opinion

St. Anthony's Hospital, January 1, 1915, to May 1, 1925.

TABLE SHOWING CASES OF ACUTE PERFORATED GASTRIC AND DUODENAL ULCERS

Case	Age	Date	Location of Ulcer	Operation	Condition 1925
1	54	1915	Gastric, anterior surface pyloric portion	Inverted Closed Drainage	Good, no gastric symptoms
2	58	1916	Duodenum	Appendicectomy	Death
3	41	1916	Duodenum, anterior right	Closure Drainage	Incisional hernia, return of ulcer symptoms.
4	33	1918	Duodenum, anterior lateral	Closure with Posterior gastro-enterostomy Drainage	Good, no distress
5	54	1919	Gastric, anterior pyloric surface	Margin of Ulcer trimmed, Ulcer Closed No drainage	Recovery
6	21	1921	Duodenum, anterior right, 2nd portion	Ulcer invaginated. Posterior no loop, gastro-enterostomy No drainage.	Recovery, no distress
7	42	1921	Duodenum, anterior surface right	Suture-posterior gastro-enterostomy drainage.	Recovery
8	56	1922	Gastric	None	Death
9	57	1923	Duodenum, near pyloric	Suture, no loop, gastro-enterostomy, no drainage	Condition satisfactory
10	41	(May) 1924	Duodenum	Suture only, drainage.	Present condition not known
11	35	(May) 1924	Duodenum, 2nd portion	Suture only, drainage.	No symptoms at present
12	41	(Dec.) 1924	Gastric	Suture only, drainage.	No symptoms
13	27	(Sep.) 1924	Duodenum	Suture only no drainage	No symptoms at present
14	52	1924	Duodenum	None	Death
15	41	1925	Duodenum	Exploration Liver Abscesses	Death
16	33	(Apr.) 1925	Duodenum	Simple suture drainage	Still in the hospital

The X-ray is useless in this condition and the laboratory is of only negative value in the early part of this disease.

The differential diagnosis is often difficult—especially is this true in those cases that give little or no history or gastric distress prior to the perforation.

The most common error in diagnosis is confusion with appendicitis. In one series of forty-nine cases of perforated ulcer, nineteen had a pre-operative diagnosis of acute appendicitis. In one fatal case of our series, the same mistake was made, the patient being operated for an acute appendix.

is that negative cultures are obtained in early cases in the majority of perforations.

It is a belief among surgeons that perforation cures ulcer, but careful investigation fails to prove this. It is true that some cases will be cured by simple suture, as two of our series. But perforation is not a guarantee of cure and we have no means of knowing which case will heal and which will not.

The treatment is of course, always surgical. The earlier the better. Delay means disaster. Early surgical intervention is desired in those acute abdominal conditions that simulate perforated ulcer.

Deaver, in following twenty-one cases of gastric and duodenal perforated ulcer, found that only two were unimproved, and these were both cases in which a gastro-enterostomy was omitted. One of these was a gastric and one a duodenal ulcer, both were cured by secondary operation.

Early operation is of the most importance. The surgical procedure is secondary. Treatment—the procedure may be one of three: 1. Closure of perforation. 2. Closure of perforation with gastro-enterostomy. 3. Radical resection of ulcer bearing area. (We may dismiss this latter from our consideration as it is unjustified in acute perforation.)

Surgeons are not agreed as to which is the best operation. Simple closure is not without its advocates, although a larger percentage of surgeons prefer closure, plus gastro-enterostomy. Wilkensky states that if gastro-enterostomy is of no, or doubtful value for non-perforating ulcer then it is of no, or doubtful value for perforated ulcer, then adds that he believes (along with Continental Surgeons) that gastro-enterostomy is not a satisfactory operation for any ulcer and advocates a resection.

Deaver aptly states that no surgeon would operate on an ulcer by simple suture in the pre-perforative stage, but some are content with simple suture as a cure after perforation.

It is agreed that gastro-enterostomy in the early cases does not increase the mortality—so this factor cannot be argued for or against a certain operation. There were no deaths in our series in which ulcer was operated either by closure or closure plus gastro-enterostomy. One case was operated for a mistaken diagnosis of appendicitis and died, the perforation not being closed. The tendency is away from peritoneal drainage, especially in early cases, although Guthrie in his questionaire found that one hundred one out of one hundred thirty-seven surgeons drained after operation for perforated ulcer. The type of drainage used should depend on time of operation and the amount of spilling of material, the delayed cases demanded ample drainage on account of peritoneal contamination. In this series drainage was used in out of twelve cases in which ulcer was operated.

Results: Gibson [9] reports seven out of twenty-eight, or 25%, duodenal ulcers operated by sulture required re-operation, but only one out of twenty-eight gastric ulcers required re-operation.

Smith [10] reports forty-one duodenal ulcers with simple closure and four required second operation.

Stewart and Barber [11] reported twenty-four cases, treated by simple closure, none requiring any other operative procedure.

Mayo [12] advocates closure with gastro-enterostomy.

Lewison believes that gastro-enterostomy should be done wherever possible.

In McCreery's [13] series twenty-two per cent of gastric perforations and sixty per cent of duodenal treated by simple suture required secondary operation.

Eugene Poole [14] observed in his series that 33 1-3% treated by a simple closure required second operation, but advocates simple closure at the time of perforation with a secondary operation if required.

We have analyzed all the cases of perforated gastric and duodenal ulcer admitt-

RESUME

Four (4) Gastric Perforations

Three	(3)	Suture only	Condition good
One	(1)	Death	Non-operative

Twelve (12) Duodenal Perforations

Four	(4)	Suture, plus gastro-enterostomy	Present condition is satisfactory
Three	(3)	Suture only	Two (2)—Present condition satisfactory One (1)—Return of ulcer symptoms
One	(1)	Suture only	Present condition unknown
One	(1)	Suture only	Patient in hospital, recent operation
Three	(3)	Deaths	(No operation (Appendix removed—wrong diagnosis (Multiple liver abscesses. Explored.

ed to St. Anthony's Hospital, Oklahoma City, Oklahoma, from January 1, 1915, to May 1, 1925. During this time there have been sixteen cases—four gastric and twelve duodenal cases. The mortality has been four cases or 25%.

Quite an interesting point has been that of all the cases operated on for ulcer there has been no post-operative death. Most of these were operated on early after the perforation, but there are a few that had gone more than twelve hours after perforation.

In those cases in which a gastro-enterostomy was added to a simple suture the patients suffered no ill effects from this added surgery.

The end results we are unable to give on all cases, but have followed up reports on three gastric and seven duodenal cases. Some of these are less than a year post-operative, one is still in the hospital. We find that three of the gastric cases, or 100% are well and are free from symptoms at this time. All of these were operated on by simple suture. Of the duodenal perforations three died. In none of these was the ulcer operated on. In one case the present condition is not known, and one case is still in the hospital. Of the remaining seven, four were treated by suture plus gastro-enterostomy, all are well with no return of symptoms. Three were treated by simple suture; two are well and free from symptoms; one has return of ulcer symptoms. That is, 33 1-3% had return of symptoms.

While these cases are not conclusive and is a small series, still they are suggestive that perforated gastric ulcers treated by suture only may cure, while in perforated duodenal ulcer—suture plus gastro-enterostomy gives better results.

Between January 1, 1915, and May 1, 1925, there have been twenty-four cases of chronic ulcer of the stomach or duodenum operated at St. Anthony's Hospital, Oklahoma City, Oklahoma. During the same period there were sixteen acute perforations of a chronic ulcer—that is, 40% of these ulcers were admitted in the perforative stage.

The mortality of perforated ulcers in this series has been four cases, or 25%. The operative mortality has been nil.

The operative mortality in the non-perforative cases has been one case, or 4%.

The question naturally arises—if the perforative cases had been operated before perforation what would have been the result?

Perforation of an ulcer after gastro- enterostomy is very, very rare, consequently we may assume that operation would have prevented perforation.

CONCLUSIONS

1. Acute perforated gastric or duodenal ulcer occurs in 7 to 15% of chronic ulcers.

2. The previous history is important.

3. It is pretty well agreed that perforation does not cure the ulcer.

4. It is universally agreed that early operation is of primary importance.

5. The operation of choice in our opinion is closure with gastro-enterostomy. This is especially true if dealing with a perforated duodenal ulcer.

6. Gastro-enterostomy does not increase post-operative mortality in early cases.

7. The peritonitis of perforated ulcer is chemical in the early hours, changing to bacterial after about 12 hours.

8. Drainage is practically always indicated except in very early cases.

9. The mortality of perforated ulcer is very high.

10. Too many ulcers are coming to the surgeon in the perforative stage.

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In the discussion of an "ulcer perforation" it must be distinctly understood in the beginning, that the subject for discussion, "Are the symptoms related entirely

to perforation, and not to the reactive peritonitis which occurs hours after the perforation and are due to a bacterial involvement of the peritoneum."

The discussion must also eliminate the perforative peritonitis symptoms that are evident in the sub-acute perforative lesion; although in some instances this type of lesion may develop suddenly into an acute process.

Perforation of a "gastro-intestinal ulcer is a condition that is always to be thought of and much to be dreaded. It is one of the most tragic episodes that occurs in surgery. The patient may be stricken in his office, in the theatre, on the train, on the street, and very often while confined as a patient in the hospital.

While attending a Clinic at Cook County Hospital at Chicago, in the fall of 1923; three specimens of perforated ulcer were shown. These patients had died while being treated in the medical wards of the institution. The physician giving the Clinic, said that, "there was an average of four deaths a month, occuring in the medical wards of the hospital." An elderly physician sitting by my side, said that, "he knew of seven recent cases in a Philadelphia Hospital." It seems to me that this is a fearful conviction of the medical man who stands by and allows a catastrophe like this to occur.

Nearly all of these patients have had symptoms for years and usually have had as many different varieties of treatment as "Heinz has pickles", and it seems to me the irony of Fate, that an individual should die from an ulcer, that has been pleading its cause and begging for relief for years.

In the ulcer perforations that I have seen, there has been always a long history of gastric disability. Suddenly, as out of a clear sky, he is stricken with an almost intolerable agony; an agony that is so gross that the individual is unable to move. The abdomen is held as if in a vise. The transverse striations of the recti can be plainly palpated. The pulse is not particularly increased, and the temperature may be normal or sub-normal. When the change occurs in the pulse and temperature, a peritonitis is developing. The proper time for operative remedies is before a bacterial invasion of the peritoneum occurs.

Statistics from various clinics very pointedly show that it is immaterial what operation is done in the early hours; recovery is the usual outcome.

As to operation: It seems to me that the best results have been obtained where the ulcer is sutured and gastro-enterostomy is done. There are some who believe that only suture of the perforation is necessary, but every surgeon who has had much experience with ulcer perforation will find it necessary to do a secondary gastro-enterostomy in quite a large percentage of these cases. It is presumed that the question of suture alone, or suture plus gastro-enterostomy will vary with the individual surgeons' experience.

Experience with gastro-enterostomy as a remedy in the ordinary non-perforating ulcer: I take it that the best statistics that have ever been shown by any man, are those of "Balfour", in which he reports that amazing number of one thousand gastro-enterostomies for ulcer after an interval of ten years. In this series, Balfour found 92% of these patients relieved of their symptoms. This inclines me to believe that a gastro-enterostomy is a good measure for an ordinary ulcer.

Therefore: If it is of so much benefit to an ordinary ulcer; is it not good logic to believe, that if the patient's physical condition is such as to stand the additional gastro-enterostomy, that it would be better to do it?

SOME DIFFICULTIES IN THE DIAGNOSIS OF THE DISEASES OF THE STOMACH, WITH SPECIAL REFERENCE TO EXTRA—GASTRIC CAUSES.*

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The importance of this subject, the very wide scope it covers, and the everydayness of its occurances in the general Practioner's office, the many mistakes in diagnosis that it may help to correct, was brought to my attention by the following case, which is the real reason for my writing this paper. I will say to start with that the diagnosis is yet unknown. I suspected a Gastric Carcinoma and advised an exploration operation for diagnosis, so referred her to a prominent Surgeon, who made a tentative diagnosis of some diseases of the Pancreas and advised an exploratory for definite diagnosis, but first called in a

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Medical Consultant who makes a tentative diagnosis of Gall-Bladder disease, but advises exploratory.

As patients husband nearly died from a Gall-Bladder operation she refused an exploratory and came back home. I was not satisfied as to any of the previous diagnoses. I called in Specialists on the Stomach and Intestines who said we were dealing with a chronic appendix and Spastic Colitis and advised removal of appendix and proper dietary management after operation.

Urine

Amt.	
Color	Amber
Appear.	Clear
React.	Acid
Sp. G.	1.025
Albumen	0
Glucose	0
Acetone	0
Diacetic	0
Indican	0
Diazo	
R. B. C.	0
W. B. C.	Occas.
Casts	0
Cyst	0
Organisms	Few
Cells	Few Squa-Epithelial

Blood

Hgh.	85%
R.B.C.	4,550,000
Index	.9
Size	Normal
Shape	"
Color	"
Blast.	0
W.B.C.	5450
P.	72
L.	25
L.M.	0
T.	2
E.	0
B.	1
Other cells	0
Platelets	Few
Parasites	0

Feces

Color	Brownish
Consist	Semi-solid
React	Neutral
Mucus	0
Pus	0
Blood	0
Fat	Small amount
Bile	XXXX
Bact.	Many
Parasites	None found

Gast. Cont.

	Ewald
Amount	40 CC.
Color	Grayish
Consist.	Limpid
Mucus	Large amount
Pus	0
Blood	0
Food	Small amount
Bact.	Many
Total acid	1
Free HCL.	0
Comb. acid	1
Lactic acid	0

On August 16-24, Patient came to office.

HISTORY OF PATIENT

Female, 39 years old, 5 ft., 6½ in. high, weight, 138 lbs. Chief complaint, pain at angle of left Scapula coming on about 4-5 A. M. every morning and wearing off about 11 A. M. She says this is the only complaint, feels fine every other way. But on closer questioning brings forth the fact of Chronic Dyspepsia, gas and palpatation, numerous hot and cold flashes, sometimes leg and headache. Periods irregular, has missed as many as 6 months, flows scant for one day, when she does menstruate. This has been her history since she started menstruating at age of 13, been married fourteen years, never been pregnant.

Past History—Negative.

Family History—Mother died of cancer of stomach.

Physical Examination—Head and neck, teeth and tonsils negative. Pulse normal B. P. 130-80.

Heart—Not enlarged, no murmurs.

Lungs—Negative.

Abdomen—No tumors or masses palpable, no points of tenderness except over McBurneys.

Female Examination—Negative, except an infantile uterus, no Leukorrhea or history inflammations.

Laboratory Findings—Wassermann-Negative.

Fluoroscopic examination and X-Ray with barium meal shows no abnormality of contour, greater curvature lying at level of umbilicus. Cap well formed, no excessive peristalsis, no indenture, stomach freely movable.

Gave Dil. HCL. bromides and whole ovary, Jan. 10, 1925. Patient has now been under my observation about 6 mos., with no improvement, only indigestion relieved, in fact has been slowly getting worse. During the time I have taken four Gastric analyses, two complete X-ray series and numerous fluoroscopic examinations, all with the same findings of normal outline of stomach and absence of HCL. with occult blood. The chief complaint of pain at angle of left Scapula is still present but has begun to radiate around like a Herpes Zoster and even penetrate straight through to left Hypochonderum which is now tender to pressure. Gastric analysis at this time shows no free HCL. total acidity 10; blood present, lactic acid and excessive mucous. X-ray pictures and fluoroscopic still negative no tumor or masses in abdomen palpable. It was at this time I insisted on consultation and advised Laporotomy for diagnosis and the consulting Surgeon and Internist looked her over as stated before and Patient came home. I then put her to bed for two weeks, as she was nervous, I thought absolute rest and relaxation might help conditions; at end of two weeks, no improvement so I called in a Gastro-Enterologist, who said "Spastic Colitis with chronic appendicitis." And advised appendectomy, but as patient refused, we continued her treatment in bed on diet and medicine for Spastic Colitis.

At end of two more weeks patient refused medicine and diet, and got up, her condition was improved. I tried to force an operation, and I told her, if for no other reason let me operate so I can make a diagnosis and complete my paper for the State Medical, but all I could get was a promise for some time this month. The variation of diagnoses on this case brought home to me the fact of the difficulties in diagnosis, when the stomach is concerned and the great possibility that the cause is outside of the stomach, for we know that the stomach is said to be the mouthpiece of disease of any portion of the body, especially the organs of the abdomen.

Mayo has told us that only 20 per cent of cases presenting gastric symptoms are due to disease of the stomach itself. That 40 per cent of the cases are due to other intra-abdominal causes and that the remaining 40 per cent have their origin outside of the abdominal cavity.

The extensive and complicated nerve supply to the stomach places it in direct or indirect communication with all of the vital organs. This is especially true as regards the gall-bladder and appendix, with which it is interconnected by nerve filaments as a result of their having similar embryonic origin. The inervation of the stomach by branches of the vagus and great sympathetic abdominal plexus is a mechanism so delicate that the slightest stimulus, as the sight or odor of food, will produce gastric secretion. Understanding this, we realize why the stomach is so often disturbed by pathological conditions outside of itself.

Everyone recognizes that pain anorexia, nausea, vomiting and gastric distress are among the very common symptoms of acute appendicitis. These symptoms in a milder but more persistent form are found in chronic and recurrent appendicitis and reflexly or otherwise give rise to the group of symptoms commonly called chronic dyspepsia.

Aaron has described pain in the epigastrium induced by pressure over the appendix in chronic appendicitis, and believes this to be due to spasm of the pylorus, this he proved by bismuth meal and X-ray.

Fenwick believes the constant excess of free Hydrochloric Acid gives rise to spasms of the Pylorus and also excites a violent gastritis. He believes that 12 per cent of the cases of hypersecretion are due to disease of the appendix, and that there is usually an increase both in quantity and acidity when active irritation of the appendix is present.

Paterson believes that the appendix influences the gastric secretion, but thinks it is due to intestinal stasis rather than pyloric spasm.

In gall-bladder trouble we may see the typical picture of chronic functional dyspepsia, fullness and discomfort after meals and especially that bane of the gastro-interologist gas in very large amounts, or in another case, a picture so like that of gastric cancer with its lack of appetite, loss of weight, etc., that even the persistent absence of occult blood and negative X-ray pictures, we often are in doubt, and only the operation definitely determines that we are dealing with a case of gall-stones or chronic cholecystitis with no local manifestations. In both these types, especially among fat women after early middle life. I find an achylia or at least an achlorhydria is the rule.

As contrasted with gastric or duodenal ulcer, an attack of gall-stones lasts a few hours, is most abrupt and severe; an acute choleocystitis lasts a few days and is less severe while a gastric ulcer is a chronic condition and is least severe.

In gall-bladder disease these referred gastric symptoms undoubtedly of vagus origin, are peculiarly interesting in that they have a definite cardiac equivalent. All the signs and symptoms of cardiac distress being quite often found in this group of cases, symptoms which entirely disappear with a realization that the heart trouble was functional and not organic, after surgery on the gall-bladder. A very wise Clinician once said, "When a patient complains of indigestion with no apparent cause, always think of the possibility of beginning myocardial insufficiency; when he or she complains of cardiac symptoms, always realize the probability that these symptoms are of gastric origin."

In making a differential diagnosis between diseases of the biliary tract and the appendix on one side and uncomplicated gastric and duodenal ulcer on the other, we can often get valuable help by remembering that in a duodenal or gastric ulcer, pure and simple, we should not have jaundice, should not have infectious headaches, should not have history of rheumatism, should have no chills or fever. If you ask yourself the question, Am I dealing with an infection or a non-infectious process? It will help make the diagnosis.

Crile has pointed out that in abdominal visceral, pain is caused by tension of the muscular wall of the part and not by irritation of the mucosa. He points out that cutting of the mucous membrane causes no pain, while stretching or tension of the walls of the organ produces pain. Thus the pain of appendicitis, cholecystitis, would depend on the overtonicity and tension of the walls of the affected parts. The character of the pain can often be differentiated if we remember that a colicky pain is relieved by pressure while an inflammatory pain is increased.

The pathology of the cord may simulate diseases of the stomach, as tabes, lues and vascular disease.

The nervous system plays a prominent role under this etiology, for the neurotic is the bug a boo of the general Practioner as well as the Specialist, and the Surgeon has operated many to return not only unimproved but with the added shock of the operation. For the so-called hysteric simulates most any gastric symptom.

The differentiation of the functional from the organic is a real art, and we should have more clinical examinations and clinical study, and not depend so much on the laboratory for diagnosis.

A new field is the Endocrine glands, what we may expect from the study of them is unknown. Would the results in the above case have been different if we had fed Ovarian Ext., to her many years ago?

The gastric picture is interesting in both acute and chronic pancreatitis, also the periodic attacks of severe nausea and vomiting in cases of market retroflexion of the uterus with no local symptoms. A very common mistake in diagnosis is the twisted pedicle of an ovarian cyst, with only upper abdominal symptoms.

Acute and chronic infectious diseases such as Typhoid fever, Pulmonary T. B. and Syphilis all have gastric manifestations.

Every one of you have had patients complaining only of gastro-intestinal sypmtoms and without cough or dysponea, chills, or sweats where physical examination shows the gastro-intestinal tract to be sound, but the lung with T. B.

Cases of intestinal parasites or pernicious anemia may fool us and we think we are dealing with a gastric cancer.

I might go almost indefinitely reciting portions of the body where the true nature of the underlying pathologic condition was entirely masked by symptoms referred exclusively to the stomach so I assume the complaining stomach to be innocent until it has been proven guilty, which will result in the search for extra as well as intragastric causes for the symptoms complained of.

SIGNIFICANCE OF PAIN IN RIGHT LOWER ABDOMEN*

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In bringing to your attention this particular symptom of abdominal pathology, I am not unmindful of the great amount of literature which has been presented under a similar title in the past. Notwithstanding this fact, however, there remains much inspiration and stimulation for such a theme due to the many patients who present themselves with right lower abdominal pain as their chief and only complaint. This paper, more or less empirical in its presentation, deals only with surgical diagnosis and is presented with the idea that by placing more emphasis on symptomaology we may more accurately diagnose and therefore more successfully treat, abdominal conditions in general. You will no doubt agree with me that there is more reason for diagnostic progress in the lower right abdomen than elsewhere. In order to limit the scope of this paper, the right lower abdomen will be defined as including the abdominal wall, the extra-peritoneal spaces and the peritoneal cavity, with such contained viscera as the terminal ilium, cæcum with its appendage, the ascending colon and the distal half of the ureter. The pelvic viscera are not included as strictly speaking they are not normally within the abdominal cavity.

I cannot agree with the expressed opinion that pain is of no significance in considering a pathological condition; if we are to succeed in obtaining the patients' viewpoint, we must believe with them that pain is all important. It will be my purpose to review briefly the anatomy of the structures which may produce pain, the physiology of the pain, the practical application of this basic and fundamental knowledge and finally present two case

* Read before the Section on Surgery and Gynecology, Annual Meeting, Oklahoma State Medical Association, Tulsa, May 12, 13, 14, 1925.

histories which typify the chronic sufferer of the right lower abdomen.

That portion of the abdominal wall under consideration is supplied by the 8th, 9th, 10th and 11th thoracic nerves, which run for the most part between the internal oblique and transversalis muscles. Branches of these nerves are found in the areolar tissue just outside of the peritoneum,—an extra-peritoneal coat. The parietal peritoneum, according to Mackensie [1], possesses no nerve supply. It is however rarely that the peritoneum is stimulated without the stimulation of the extra-peritoneal coat, so closely applied to it and which is extremely sensitive to pain. The sensory nerve supply to the viscera under consideration is thus the sympathetic fibers which originate in the plexuses of Auerbach and Meissner. Neither the vagus or the splanchnics are directly connected with these viscera. The sympathetic fibers lie in the mesentery with the blood supply and as afferent fibers reach first the superior mesenteric plexus since this portion of the alimentary canal is supplied by the superior mesenteric artery. From the superior mesenteric plexus there is a direct connection with the coeliac plexus in the epigastrim. The ureter is supplied by the spermatic plexus of the sympathetic which reaches the coeliac thru the abdominal plexus.

Little need be said in regard to the anatomical structure of the viscera of the right lower abdomen. Mention should be made, however, of the ileo sphincter which is one of a series of gastro-intestinal sphincters all controlled by the efferent sympathetic fibers and first described by Elliott and Smith [2]. This ileo sphincter is located just proximal to the ileo-cœcal valve and stimulation of sympathetic fibers relaxes this sphincter with the others in the series. The cæcum may be one of three types; foetal, wherein it is symmetrical and conical, the vermiform process leading off from the apex of the cone; infantile, where the lateral wall is most prominent, the process coming off more abruptly, and finally the adult or so-called normal type which shows the lateral wall projected downward to form the true end of the cul-de-sac, the medial wall dragging behind in the process of development. The cæcum is at once the most dependent and most freely movable portion of the large bowel. The right ureter lies upon the psoas muscle and in the sensitive extra-peritoneal areolar tissue, extending downward and medially. It thus passes medial to the cæcum on its way to the side wall of the bladder.

Pain is produced by stimulation of nerves which have the ability to transmit stimuli to centrally located receptive areas. Stimulation is not confined to the terminal fibers but may be produced anywhere thruout the course of the nerve. Not only the cerebro-spinal nerves but also the afferent fibers of the sympathic system are capable of stimulation and conduction of painful impulses. The normal viscus does not give a sensation specifically but adds to the general feeling of well being. As a result of disturbed structure or functioning of a viscus we have the initial response thru the sympathetic system. The end plexuses are stimulated and sensations are continually sent to the plexus higher in the chain where they are stored. In the case of these particular viscera, the sensations are stored in the superior mesenteric and aortic (for the ureter) plexuses until these higher plexuses become so sensitive that the stimuli break thru and reach the coeliac. It may be said that the superior mesenteric is a mirror reflecting all the pain producing conditions within the right lower abdomen and in turn focusing its rays on to the coeliac plexus. Stimuli passing thru these plexuses reach the spinal cord thru communicating rami and some are then propogated out thru the spinal nerves in the production of the "viscero-sensory reflex". One must distinguish between the two different types of pain,—the somatic pain described by Ross [3] which concerns the cerebro-spinal nerves and the visceral pain confined to the viscus and transmitted by the sympathetics and brought into consciousness without propogation. It is common for the patient to describe visceral pain as "deep" or as "middle" pain. Thus the physiology of the production of pain is clear and definite; the perception and interpretation of pain on the other hand, is as different as the individual who experiences it. Psychical causes such as bereavement, anxiety, frequently some surgical procedure, will bring a pain into consciousness without appreciable change in the pathology. It must be realized that with increasing civilization, the threshold to pain becomes lowered. What would be of no consequence to one of a sanguine disposition, would be extremely distressing or unbearable to the neurotic.

In regard to pain of the abdominal parietes, the common causes of trauma and

other external factors need only be mentioned. It should not be forgotten, however, that posterior root disease and tuberculosis of the spine may produce typical pain due to the involvement of the spinal nerve trunks at their source. Traction on the parietal peritoneum from any cause, direct or indirect, by pull on the closely applied extra-peritoneal layer will produce somatic pain. Should the serous tissue alone be irritated, there is no sensation. Within the abdomen we may have dragging down and tension on surrounding tissues which will produce painful sensations. As an instance, the dragging of the mesentery of the dependent ilium and cæcum, due to ilio-cœcal stagnation, makes ligaments of suspension of the nerve fibers running between the layers of the mesentery and is a source of persistent painful sitmulation. The ureter because of its association to the sensitive extra-peritoneal areolar tissue will, if dragging is permitted, as it is in certain degrees of movable kidney, cause severe pain involving, in this instance only spinal nerves. A weighty boggy cæcum may indirectly cause tension on the ureter.

From this consideration of the spinal nerves in the production of pain in the right lower abdomen, let us now pass to the role of the sympathetic nerves. It may be laid down as a dictum, since it has been definitely proven, [4] that there is only one cause for stimulation of afferent fibers and therefore one cause for pain in hollow viscera and that is distention. Such distention is due to an increase in intravisceral pressure and may or may not be accompanied by smooth muscle spasm. As an instance of distention we have that produced by the fluids introduced in an enema,—pain is thus produced which is instantly relieved by the explusion of fluid and the relief of the tension. All of the viscera located in the lower right abdomen are hollow viscera, hence we may deduct the fact that intra-abdominal pain in such an area is due to the distention of the viscus involved. Such distention may or may not be accompanied by inflammation as a causative factor. It has been very clearly pointed out that pain accompanies the distention above ureteral stricture without the presence of active inflammation. The terminal ilium, cæcum with its appendage, and the ascending colon likewise may be chronically distended due to a variety of causes and may produce the same pain interpreted and propogated in the same manner. A generalized sympathetic stimulation will inhibit sphincter action and peristaltic waves and thereby cause cæcal delay and stagnation from physiological causes only. It should be emphasized finally that these various causes may exist in a latent form until susceptibility becomes exaggerated when they are brought into conscious painful conditions and remain to the great distress and discomfort of the individual.

There follows two case histories which are typical of this condition:

1. Mrs. H., white female, age 33, complains of pain in the right lower abdomen of several years duration. It is constant and aching in character, at times severe enough to incapacitate her. It bears no relation to meals,—at times is propogated to the epigastrium and usually relieved by lying down. She suffered from constipation as long as she can remember. Has had three operative attempts to relieve the pain: appendectomy, salpingo-oophorectomy and hysterectomy. Following each operation, she experienced relief for a varying length of time. She is extremely nervous, introspective, and irritable. Examination shows a very poorly nourished young lady with normal pulse, temperature and respirations. The blood-pressure is 115 over 80. Abdomen shows scars of former operations, costal angle is hyposthenic, abdominal walls flaccid and poorly developed. There is moderate distention. Diffuse tenderness is present over the abdomen,—especially marked in the right lower. There is point tenderness over both Morses and McBurneys points. There is palable gurgling or borborygmus over the cæcum. The X-ray shows paosis of the transverse colon and stomach with coecal delay and stasis.

2. Mr. C., white male, age 44, has had lower right adbominal pain for ten years gradually growing worse. Eight years ago he was advised to have the appendix removed but refused. At times the pain becomes very severe and is propogated to the epigastrim. There is no relation to meals but the pain is aggravted by constipation from which he suffers constantly. There is no nausea or vomiting. He worries a great deal about business and family affairs and is constantly under high nervous tension. He has lost twenty pounds in weight the past three months following removal of tonsils. Examination shows a sthenic type of costal arch,—flaccid abdominal walls without scars. There is

marked local tenderness over the cæcum with borborygmus and also epigastric tenderness. The descending colon is palpable, rope-like and tender,—there is tenderness over superior and inferior mesenteric plexuses. X-ray done at Rochester, Minn., showed marked dilitation of the caecum with stasis.

In discussing the first case one is tempted to paraphrase a text of our brethren of the cloth and say, "By their scars, ye shall know them." Approximately 80% have scars of McBurney, right or midrectus incision. The second case had repeatedly declined operation, altho there were many opportunities to submit. Both had epigastric tenderness due to the fact, I believe, that the superior mesenteric plexus had become so irritated by repeated assaults that the stimuli had broken thru to the coeliac plexus. Both individuals had a lowered pain threshold, were inclined to be introspective and hypochondrical. The first patient showed many evidences of infantilism and I believe she had an infantile type of cæcum.

In conclusion, let me say that pain is purposive; either visceral or somatic pain excites a reaction which is also purposive. Pain should be regarded as a friend of both the patient and the diagnostician, not as an enemy to be entirely annihilated by the loaded syringe. The Utopian idea of medicine is that there shall be no more pain, yet that mythical state shall never be reached unless we can more accurately and therefore more effectively trace the origin of pain. He who attempts to understand pain without taking into consideration the sympathetic supply of the viscera, will find many inexplicable conditions confronting him, and the mysteries of diagnosis ever increasing. Let us depart from the usual summarizing and allow me, please, to tell at least one reason for this paper. I am first of all not quite convinced that appendicitis per se does not cause pain in the right lower abdomen. I am not convinced of the clinical entity of chronic appendicitis. I was inquisitive to determine whether or no a paper on pain in this portion of the abdomen could be prepared without mentioning appendicitis. Taking a plan from noted generals in battle who start a small offensive to detract attention from a larger, much more important one, I endeavored to emphasize the conditions in the right lower abdomen which might cause pain, without reference to the murderous term of chronic appendicitis.

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THE ROENTGEN DIAGNOSIS OF DUODENAL ULCER*

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Duodenal ulcer, though long recognized as a clinical and pathological entity, has received its proper attention and careful differential diagnosis only within the last decade. The earlier investigations were conducted in the clinics of Europe, and hence this group of pioneer observers has been styled the Continental School. To such men as Haudek, Haenisch, Reider, Holznecht, Rosenthal, etc., belongs the credit for the first definite and recognized work in this most interesting field of diagnosis. Almost of necessity, however, due to their large clinics, lack of adequate funds, and imperfect apparatus, they gradually built up a series of functional signs, observed with the fluoroscope, which is today known as the indirect method of examination.

But the scientific medical world awaited, as it has in many other lines of endeavor, the brain child of an American genius to visualize the actual ulcer area itself. The brilliant researches of Lewis Gregory Cole of New York established on a firm and enduring basis, by means of "serial radiography", the definite and localized character of this all too frequent lesion. Cole's work was initiated in the years just prior to the World War; and although he shows a tendency to minimize, if not indeed to practically ignore, the fluoroscopic findings in the diagnosis of duodenal ulcer, yet his conclusions, in the main, have stood the acid test of ruthless investigation by capable roentgenologists thruout the world. Today the direct or American School champions the generally accepted standard of diagnostic excellence.

Duodenal ulcer has been demonstrated in late years to be of much more frequent occurence than was formerly supposed and

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this definite knowledge has been largely due to the visualizing of this condition by the roentgenologist. N. J. Nessa of Sioux Falls, S. D., reports a series of 30,000 cases of gastro-enteric pathology, gleaned from recent medical literature of the United States, among which he lists 15% of proven duodenal ulcers. Richard A. Rendich of Brooklyn, N. Y., quotes a compilation of 1916 positive gastro-intestinal diagnoses by means of the Roentgen ray from the files of Bellevue Hospital, over a period of three years, in which 314 duodenal ulcers were noted.

Before entering upon a discussion of the pathology involved in duodenal ulcer, it may be well to glance hastily at a few anatomical points of interest. As is familiar to all, this part of the small intestine is divided arbitrarily into a triad; namely, the first portion, the cap, or bulbus duodeni, the second or descending portion, and the third or the horizontal portion. The first of these divisions is a rather unique organ, due to the fact that it is the transition point from stomach to intestine and is known to possess characteristics of both. The duodenal mucous membrane is smooth on account of the absence of valvulæ conniventes and its musculature is, in a general way, but the direct continuation of that of the stomach. The thinner longitudinal muscle bundles form the outer coat, through which in many locations the heavier circular fibres may be made out and its greatest density is found at the lesser curvature border of the cap, a fact that will be referred to again when considering the mechanism of the niche deformity. The bulbus duodeni is supported by the duodeno-hepatic ligament and into the composition of this structure some of the longitudinal muscle fibres of the duodenum enter.

In order to make a satisfactory examination of any portion of the gastro-intestinal tract, a suitable contrast medium is the roentgenologist's first essential. Many and varied mixtures were employed before a uniformly acceptable meal was hit upon. The earlier workers used a combination of bismuth and cooked cereal, later substituting barium sulphate for the first ingredient. It was found, however, after repeated trials, that this composition did not round out the duodenal cap sufficiently to make an accurate observation of its contour. Hence these investigators sought a liquid base for their contrast medium and eventually arrived at our generally employed malted milk or buttermilk-barium sulphate-bicarbonate of soda-acacia meal. The addition of raspberry or vanilla flavoring, advocated by some workers, I have found has a tendency to nauseate a fasting patient and hence I do not include it among the ingredients.

Employing the above mentioned liquid meal, the normal duodenal cap is seen to be of conical or bee-hive shape, its basal or pyloric border straight and its lesser and greater curvature borders slightly convex and smooth in outline. It is the finding of a constant deformity of this bulbus duodeni which we shall stress in our later discussion. The second and third portions of the duodenum present only an ill-defined, feathery flecking of barium particles in the average case, with no workable outline of the walls of the intestine.

It is very essential that the gastro-intestinal tract under examination be free of food and fecal matter, hence the roentgenologist working in a hospital has a decided advantage over the office practitioner, whose control of his patient is more or less limited and who lacks the skilled assistance so necessary for the proper preliminary preparation of the subject for diagnosis. There is much discussion and wide divergence of opinion as to the necessity for catharsis. My instructions contain an order of castor oil or compound licorice powder to be taken the day before and a cleansing enema the morning of examination. The morning meal is of course omitted. This routine, if intelligently complied with, usually gives me a clear field for operations.

There is radical disagreement among roentgenologists as to the relative importance of screen and plate in the diagnosis of duodenal ulcer. Some, aligning themselves with the Continental School, place the fluoroscopic examination and indirect signs to the forefront, basing their findings on these, and considering the serial plates as of only secondary or confirmatory importance. While others, following Cole, George, and many of the American School, would consider the serial plates showing the defects of the cap as sufficient evidence and employ the fluoroscope only as a necessary, tho often misleading, adjuvant. A third group, the outstanding member of which is Carmen of the Mayo Clinic, would compromise with these two more radical viewpoints, and while assigning to the pictured deformity in serial

plates its justly preeminent station, nevertheless consider the indirect signs as of the utmost value in a proper and conclusive diagnosis of the pathology present. From my rather limited experience I should prefer to take my stand with this last group, since I find that I can ill afford to scorn any legitimate aid in this very difficult and ofttimes confusing field of diagnosis.

Having administered the barium by mouth, the examiner allows sufficient time for the full and vigorous peristaltic waves of the stomach to become the established order, and when this has occurred, the bulb of the normal duodenum should be outlined as a smooth walled cone, whose base is formed by the pylorus. According to Alvarez, "either simultaneously with, or shortly after, the arrival of a gastric peristaltic wave at the pyloric line, there is a strong tonus contraction in the duodenum. This observation may throw some light on the mode of development of the pain in duodenal ulcer, because we now have every reason to believe that hunger contractions in the stomach will produce similar contractions in the duodenum. It is also interesting to note that the peristalic rushes which carry the food down the second portion of the small bowel almost always have their origin in tone changes in the duodenal cap."

Our main endeavor is to adequately visualize the duodenal bulb and this is attempted in the vertical position, first using the upright fluoroscope. If there seems to be an irregularity in the contour of the cap, pressure with the gloved hand is resorted to in order to force the contrast mixture to round out the first portion. But the most satisfactory view is often obtained with the patient in the horizontal position, rotated slightly to the right and lying face down on the table. Rotation in various directions seldom fails to bring the cap into view at some angle. Nevertheless, in very fleshy individuals or those with pronounced steer-horn stomachs with the duodenum occupying a posterior position, I have found great difficulty and, in some cases, have been wholly unable to demonstrate tne cap under the fluoroscope. After observing the secondary functional signs which may be present, a series of plates is made in which I endeavor to show the contour of the bulb at short intervals. Any persistent deformity is usually first observed on the fluoroscopic screen and then confirmed on the plates.

A very conservative estimate places nine tenths of all duodenal ulcers in the first portion or cap region and the selective site would seem to be the lesser curvature border. The other tenth occur as single or multiple ulcerations of variable size and structure and may be situated anywhere in the second or third portion of the duodenum. These latter, however, are diagnosed more by inference and indirect signs, since the whole length of the duodenum becomes evident only when the barium mixture flows freely from a gaping pylorus or when there is an artificial or real organic obstruction at the duodenojejunal junction.

The most characteristic and dependable sign of duodenal ulcer is now conceded by the majority of roentgenologists to be deformity of the bulbus duodeni and the niche type is claimed to be the most frequent form of such irregularity, (66%-75%). Its formation is due to the filling out of the ulcer crater with the contrast mixture and, when demonstrated as a constant defect on the bulbar shadow, may be considered as diagnostic. Occurring as it usually does on the lesser curvature border of the cap, it is produced by the tightening or contraction of the thick bundles of longitudinal muscle fibres in this location, excited by inflammatory changes in the ulcer area. The circular coat is thus compressed and results in an elevation or puckering tissues surrounding the crater. This process throws the ulcer, as seen in the niche defect, into relief and we obtain a profile view. The niche is encountered in all forms of ulcer from the simple mucosal type to the indurated and calloused variety. Hence the amount of distortion of the bulb by this lesion may be out of all proportion to the findings at operation and so it is unsafe to predict the size of an ulcer from the Roentgen appearance.

Other marked deformities include a general distortion of the bulbar shadow, due either to cicatrical contractures or spasmodic manifestations, irregularities of the basal border, various species of incisurae, either unilateral or bilateral, occurring most frequently opposite the ulcer site on the greater curvature border.

In cases where the diagnosis is doubtful, we may rule out the purely functional and spasmodic deformities by the administration of 20-25 drops of tincture of belladonna three time a day to physiological effect. This will produce such relaxtion that at the second examination either the spas-

tic defects will have entirely disappeared or so altered their former appearance as to demonstrate their nature. The point to be emphasized is that the size or shape of the defect is of minor importance as compared with its constancy and similarity in outline in serial plates.

It was at first advanced as the main objection to accepting the bulbar deformity as a major sign of duodenal ulcer that there were many extrinsic causes for the same appearance. While this fact cannot be denied and may lead to quite a few erroneous conclusions yet there are many factors which will aid a careful examiner to make these failures a minimum. Extensive adhesions about the cap from a cholecystitis or reflex spasm from an appendix chronically inflamed are perhaps the most frequent extraneous causes. Also the enlarged gall bladder may produce a broad, concave, smooth-walled defect in the upper portion of the bulb.

Of the indirect signs of duodenal ulcer, gastric hypertonus, hyperperistalsis, and hypermotility are the most commonly noted. These are encountered in other conditions, it is true, but they are so often an accompaniment of this lesion, that they are a distinct dagnostic aid. In the obstructive cases, due to marked cicatricial contraction, even despite the violent peristalsis, there remains a six-hour retention in the stomach. In the non-obstructive type, an abnormally rapid evacuation of the stomach contents is usually associated with the other members of the "hyper" group and at the six-hour examination we may find that the barium mixture has made the circuit of the colon. Tenderness on pressure over the duodenum is given a very unimportant position in the catalogue of signs of ulcer, as any inflammatory condition in the upper quadrant on the right may produce such a symptom. Tho we do not attempt to minimize the diagnostic importance of deformity of the bulbus duodeni, yet it does seem to us that the indirect, functional signs should not be cast aside as valueless, since many times they give pertinent data as to the intensity of the pathological process and are often an index to the necessity for operative procedures.

Have we statistics to prove the value of roentgenology in the diagnosis of duodenal ulcer? The answer may be found in the record of one of our foremost clinics, where a great volume of this work is carefully checked with operative findings. Carmen presents a series of 523 Roentgen diagnoses of duodenal ulcer with four hundred and ninety-nine of them confirmed by the surgeon. In the same period, out of 544 negative diagnoses, 512 were free of duodenal ulcer at operation. Prior to 1920, some 4,000 cases of duodenal ulcer had been proven as correct by operative procedure, all of them having been previously diagnosed by the roentgenologist.

Schinz at the Central Roentgen Institute of Zurich, Switzerland, has reported 66 carefully checked Roentgen diagnoses followed to operation and states that the number accurately localized as well was astonishingly large. His enthusiastic conclusion seems to be that no ulcer of any size should escape detection.

Foerster, on the other hand, reminds us of the fallibility of our most prized diagnostic procedures, by quoting in one of the European journals, a series of nine cases, giving by history, clinical and roentgenological observation all the classical signs of duodenal ulcer, not one of which at operation presented such a lesion. Adhesions of an extensive nature were responsible for the errors in diagnosis.

In conclusion, Carmen, in his most excellent volume on The Roentgen Diagnosis of Diseases of the Alimentary Canal, has very tersely and satisfactorily expressed the proper attitude of the conscientious roentgenologist toward an interpretation of his findings. He says, "Failure to discover a lesion by the Roentgen ray is often pardonable, but the subjection of a patient to needless operation on trivial Roentgen evidence alone is not readily forgiven by those concerned. To sum up, the essentials for satisfactory Roentgen diagnosis are faithfulness to an orderly routine, careful observation, the use of antispasmodics and re-examination in doubtful cases, cautious interpretation, and last, but not least, a willingness to admit, whenever it is true, that the findings are inconclusive."

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Discussion: H. Lee Farris, M.D., Tulsa.

Mr. Chairman and Gentlemen: I desire to congratulate Dr. Venable on his splendid paper, which shows very clearly he is master of the subject. It is so concise and superbly written as to completely cover this difficult subject. It seems to me that all points were so thoroughly dwelt upon as to leave practically no room for discussion. However, I heartily concur in all that he has said and will stress only three or four points. Like Dr. Venable, I am not an adherent to the screen alone, nor likewise to the serial plates. I am positive that too frequently Roentgenologists are too prone to rely upon one or another method of examination when we should always use every available means to clinch our diagnosis. The late Walter Mills of St. Louis, and Carmen, also, used to always screen first and sketch what they saw; then to verify if possible, by serial plates. Many, many times I have watched them and have been agreeably surprised to see how frequently they proved their findings; therefore, I heartily agree with Dr. Venable that we should never rely on any one method of procedure.

In conclusion, I desire to impress upon you the following thoughts; which are in substance those of Dr. Diamond of New York:

(a). Ulcer always produces morphological changes in the contour of the duodenum.

(b). That only very rarely does the duodenum appear normal in the presence of an ulcer—unless there is absence of a spasm.

(c). Roentgen Ray frequently discloses a niche which the surgeon cannot palpate and a negative surgical diagnosis is not justifiable unless the duodenum is opened and inspected; therefore, the X-ray diagnosis often supersedes surgical exploration.

(d). In interpreting duodenal deformties we must always try to differentiate exactly, if possible (that is) as to whether it be a scar, a niche or an infiltration.

(e). Always remember the pathological entities (adhesions, etc.), also their manners and behavior under the Ray.

DALLAS WILL ENTERTAIN THE SOUTHERN MEDICAL ASSOCIATION IN NOVEMBER.

A warm invitation is being extended to the doctors of the South to attend the annual meeting this fall, and preparations are being made to entertain between four and five thousand. Already, 1500 rooms in the best hotels have been set aside for this purpose, and it is estimated that more will be available.

Dallas has all the chief requirements for a successful convention city; ample hotels and auditorium, easy accessibility, facilities for entertainment and diversions, coupled with wholehearted hospitality on the part of the citizenship. It is not only a medical center of importance, but a city of interest and opportunity.

Ten trunk line steam railroads serve Dallas, with 100 passenger trains daily in and out of the $6,500,000 Union Terminal Station. 258 interurban trains leave the $1,000,000 electric interurban station daily. Dallas is 16 hours by rail from Kansas City, 18 hours from St. Louis, 27 hours from Chicago or Cincinnati, and 43 hours to New York.

For those who wish to use the automobile in attending the S. M. A. convention. Dallas is located on five transcontinental highways—Bankhead, Meridian, King of Trails, Dallas-Canadian-Denver, and the Dixie Overland. These highway organizations assure the tourist of well kept roads. In Dallas County alone 1000 miles of surfaced highways, and a tourist comp and center of highway information are available also.

Dallas has a number of strong clubs, splendidly housed, such as the Dallas Athletic Club, University Club, City Club, a number of fine golf clubs, and all the leading service organizations, such as Rotary, Lions, Kiwanis are represented here—all are most hospitable in the entertainment of visitors.

Restaurants, either connected with hotels or independent, are numerous and of a generally high standard. Some of the highest priced chefs in the nation are here. You can get meals with a Western flavor, Mexican dishes, Chinese dishes or old fashioned Southern cooking. All the year truck gardens and farms are producing in some parts of Texas, and this coupled with proximity to packing houses, poultry farms and orchards, tends to keep food prices reasonable.

Dallas has 37 theatres, with a combined seating capacity of 28,000. These include summer and winter stock companies, many good road shows during the season, high class vaudeville and motion picture houses, and the Little Theatre which was twice awarded the Belasco Prize. There are theatres costing as much as $2,000,000 and seating as many as 3,000 persons.

Dallas' climate as a whole is pleasant and invigorating, without severe extremes, and November in Texas as a rule is crisp and clear, ideal for travel and for outdoor sports.

Through the medium of this Journal, in the later issues, date on the Hospital and Clinical facilities of the Convention City will be given, meanwhile, the medical profession of Dallas and of Texas, invites you to plan to attend the Southern Medical Association Convention this fall.

CURTIS ROSSER, M.D.,
For the Publicity Committee.

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EDITORIAL

REDUCING PHYSICIANS' INCOME TAX.

In computing physicians' income taxes, the Commissioner of Internal Revenue, for several years has refused to deduct expenses incurred in traveling to attend meetings of medical organizations. This has been an attitude and position despite a statement in the law that "In computing net income there shall be allowed as deductions (1) All ordinary and necessary expenses paid or incurred during the taxable year in carrying on any trade or business, including . . . . traveling expenses (including the entire amount expended for meals and lodging) while away from home in the pursuit of a trade or business." One Deputy State Commissioner has classified the expenses as "personal expenses".

When the Harrison Anti-Narcotic Act was enacted, physicians were required to pay an annual tax of $1.00 for a license. The Act was passed through Congress at the time under the guise of a "Revenue" producing measure, though it is well known, and has been since declared by repeated decisions of the United States Supreme Court, that the end in view, while ostensibly revenue producing, was in reality corrective or preventive legislation, with moral and regulatory phases. The general opinion is that the law was passed by Congress in that guise to enable them to actually invade further than they already had, the strict rights and domains of the states, which otherwise could not be done. The fee of $1.00 was attached merely to cover the cost of administration. Under the stress of war's demand, a further addition of two dollars annually was made, making the fee $3.00, where it now stands today, one of the few unreduced and certainly unjust taxes. Unreduced simply because it has been too small a matter to haggle over by any individual, notwithstanding the violation of a principle of fair dealing, the physician already overburdened with all sorts of petty interferences pays and pays and pays. Expenses incirent to improving one's ability to combat disease are as essential, as important and farreaching as the necessity to replace a broken thermometer, buy gas and oil for the car, pay office and telephone rent, hire collectors or engender any other well-known deductable expense. The Government is now and has been either entirely abolishing or greatly reducing all war time taxation measures, why not spread the matter equitably and give the physician the benefit as well as the plutocratic rich or the soda fountain habitue?

The matter of further reduction is in the hands of the Ways and Means Committee of Congress, Honorable William R. Green, Chairman. Every Oklahoma physician who believes in fair usages should ask his Congressman to at once call Mr. Green's attention to the matter and ask him to suggest that the unnecessary tax feature of the Harrison Law be eliminated and that a liberal provision be written into the In-

come Tax Law, providing for allowance of such expenses as above noted, which clearly fall into the class which should be eliminated. The Committee is now considering the matter, so our action should be taken at once. Write your Congressman, or better still ask him personally.

THE WARNING RUMBLINGS OF SMALLPOX.

As we have previously noted, smallpox, like most of the contagious diseases seems to have cycles of mildness followed by outbreaks of severity of more or less gravity. This very habit of mildness scores heavily eventually upon unsuspecting, careless people and communities. For years the disease has been so mild in some localities that no one paid much attention to it. Now the pendulum seems to be swinging again to the danger point, in some centers it has already created unbelievable havoc until the actual reports are read. In the United States within the last few months many epidemics have occured where the toll of life was unusually heavy, this despite the fact that men who have observed many epidemics and thousands of cases have not seen a death from the disease. The Kansas City and Denver epidemics alone are evidences of this peculiarity.

Compiled reports from all over the world show that it is now more prevalent than ever before, one or two countries of Continental Europe and Egypt are about the only exceptions to widely scattered epidemics. As is well known the treatment of the disease is not satisfactory, quarantine is more than worthless and useless on account of lending a feeling of false security, while preventive vaccination is the most satisfactory and sensible of all measures. Twenty-four cases with 9 deaths in one small town in New Jersey seems to warrant considerable attention to the matter. We may soon expect a reversion of type in Oklahoma.

Editorial Notes—Personal and General

DR. H. M. WHEELER, Helena, and Miss Ethel McCune announce their marriage on July 2, at Kansas City. Dr. and Mrs. Wheeler have been at home since July 15th.

DR. H. H. WILSON, Frederick, and the tourist party accompanying him are in San Francisco, and plan to visit a few more points of interest on the Coast before returning home.

DR. O. R. GREGG, Pawhuska, is taking a special course in urology at Kansas City.

DR. G. A. COMP, Manitou, who was seriously ill at his home, is reported as improving.

DR. W. A. HOWARD Chelsea, attended the O. R.C. camp at Fort Snelling, Minn., in July.

DR. E. B. THOMPSON, Duncan, returned recently from an extended trip through the Western states.

DR. J. C. ROSS, Woodward, has removed to Los Angeles, California, where he will establish his practice.

DR. JAMES D. OSBORN, JR., Frederick, has returned from Chicago, where he has been taking some special work.

DR. and Mrs G. E. STANBRO, have returned from the European trip with the Interstate Post Graduate Assembly.

DR. and MRS. T. F. GROSS, and sons, returned recently from a month's motor trip to points in the Southern states.

Dr. McLAIN ROGERS, Clinton, announces the association of Dr. Charles J. Alexander with the Clinton Hospital staff.

DR. LEONARD G. WASHINGTON, Tulsa, who was reported as having slashed his throat and wrists on a train en route to St. Louis, is still in a critical condition in the City Hospital, St. Louis. Dr. Washington is believed to be suffering from anemia.

DR. CHARLES THOMAS WHITE, Tonkawa, died July 20, 1925, of neuritis, after having suffered with disease for over two years. Dr. White was born in Illinois in 1856 and is a graduate of Rush Medical College in 1893, and Missouri Medical College in 1899. He practiced in Oklahoma for 35 years.

DR. A. L. STOCKS, Muskogee, as a member of the Committee on Public Policy and Instruction of Public, delivered an address to teachers taking the summer course at Northeastern Normal School, Tahlequah. There were some 1200 teachers present. The subject of the Doctor's lecture was "The Relationship of Scientific Medicine to the Community". The ideals and purposes of organized medicine were presented forcibly and entertainingly and were very well received.

BOOK REVIEWS

MODERN SURGERY, General and Operative, by J. Chalmers Da Costa, M.D., LL.D., F.A.C.S Samuel D. Gross Professor of Surgery, Jefferson Medical College, Philadelphia, Ninth Edition, Revised and Reset. Octavo of 1527 pages with 1200 illustrations, some in colors. Philadelphia and London: W. B. Saunders Company, 1925 cloth, $10.00 net.

BOARD OF MEDICAL EXAMINERS—STATE OF OKLAHOMA

The table below gives the names and other date concerning the Doctors who appeared before the Board at their meeting June 16 and 17, 1925, and were licensed under date of June 20, 1925:

Name	Address	College	Date of Grad.	License No.	How Licensed
Ira Ki Cummings	Ponca City, Okla.	Univ of Texas.	1924	3424	Reciprocity Texas
Will Mack Majors	Lafe, Ark.	Memphis Hosp. Med.	1912	3425	Reciprocity Ark.
Chas. Robt. Silverthorne	Woodward, Okla.	Univ. of South, Sewanee	1899	3426	Reciprocity Kans.
Paul R. Nussbaum	St. Louis, Mo.	St. Louis U. Med. School	1924	3427	Reciprocity Mo.
William Wynne Hicks	Okmulgee, Okla	Univ. of Virginia.	1920	3428	Reciprocity Miss.
Omar Autry Kirby	Marietta, Okla.	Univ. of Louisville	1920	3429	Reciprocity Texas
Chas Jerome Alexander	St. Louis, Mo.	Washington Univ. Med.	1924	3430	Reciprocity Mo.
Wm. Dewey DeLay	Durant, Okla.	Vanderbilt Med.	1923	3431	Reciprocity Tenn.
Anon Warren Roberts	Allen, Okla.	Baylor Med. College	1924	3432	Reciprocity Texas
Herbert R. Goshorn	Maysville, Okla.	Univ. Med. Col , K. C.	1906	3433	Reciprocity Kans.
Tolar R. White	Kingman, Ariz.	Memphis Hosp. Med. Col.	1901	3434	Re-registration
Wm S Woodford	Douthat, Okla.	K. C. Med. Col.	1901	3435	Re-registration
Grady F. Mathews	Oklahoma City, Okla.	Oklahoma University	1925	3436	Examination
Arthur F. Hanson	Chickasha, Okla.	Oklahoma University	1925	3437	Examination
Wm. Bronnie Wild	Norman, Okla.	Oklahoma University	1925	3538	Examination
Emery E. Ailing	Guthrie, Okla.	Oklahoma University	1925	3439	Examination
J. Wendell Mercer	Lambert, Okla.	Oklahoma University	1925	3540	Examination
Allen C. Kramer	Oklahoma City, Okla.	Oklahoma University	1925	3441	Examination
John H. Robinson	Oklahoma City, Okla.	Oklahoma University	1925	3442	Examination
Aaron S. Price	Oklahoma City, Okla.	Univ. of Louisville	1924	3443	Examination
Wm. O. Smith	Oklahoma City, Okla.	Oklahoma University	1925	3444	Examination
Byrum B. Brown	Davis, Okla.	Oklahoma University	1925	3445	Examination
Forrest Hale	Cherokee, Okla.	Oklahoma University	1925	3446	Examination
Van Dolph Herrington	Pryor, Okla	Oklahoma University	1925	3447	Examination
Joseph Elmer Kanatser	Shawnee, Okla.	Northwestern Med.	1923	3448	Examination
Clarence R. Bernard	Nowata, Okla.	Oklahoma University	1925	3449	Examination
Byron R. Gayman	Britton, Okla.	Oklahoma University	1924	3450	Examination
Julius Ernest Levick	Van Couver, B. C.	University of Manitoba	1924	3451	Examination
Laile G. Neal	Oklahoma City, Okla.	Oklahoma University	1925	3452	Examination
Loren V. Baker	Sentinel, Okla.	Oklahoma University	1925	3453	Examination
Alvin Gorby	Oklahoma City, Okla.	Oklahoma University	1925	3454	Examination
Andrew F. Giesen	Oklahoma City, Okla.	Oklahoma University	1925	3455	Examination
Forrest A Harrison	Ardmore, Okla.	Washington Univ	1924	3456	Examination
Donald W. Branham	Oklahoma City, Okla.	Oklahoma University	1925	3457	Examination
Emery E. Beechwood	Oklahoma City, Okla.	Creighton Med. Col.	1924	3458	Examination
Claude H. Miears	Oklahoma City, Okla.	Oklahoma University	1925	3459	Examination
Cleo F. Cailey	Oklahoma City, Okla.	Oklahoma University	1925	3460	Examination
Harry W. Steinig	Oklahoma City, Okla.	Oklahoma University	1925	3461	Examination
Robert L. Hamilton	Westville, Okla.	Oklahoma University	1925	3462	Examination
Jesse J Hoover	Oklahoma City, Okla.	Oklahoma University	1925	3463	Examination
Lester F. Smith	Oklahoma City, Okla.	Oklahoma University	1925	3464	Examination
James S. Perry	Tulsa, Okla.	Oklahoma University	1925	3465	Examination
Chas R. Rayburn	Norman, Okla.	Oklahoma University	1925	3466	Examination
Flora A. Wright	Oklahoma City, Okla.	Oklahoma University	1925	3467	Examination
Franklin Jelsma	Oklahoma City, Okla.	Oklahoma University	1925	3468	Examination
Robert C. Sullivan	Ardmore, Okla.	Vanderbilt Med.	1922	3469	Reciprocity Tenn.

DaCosta's surgery has so long been accepted as authoritative, the work covers such a wide range of subjects that any sort of review must necessarily reduce itself to generalities. For, if we consider the good things the work contains, pages would be written with much yet left of great value. Surgical bacteriology, asepsis and bandaging are excluded, with the advice from the author that they should be sought in special works now dealing with them, or in large text-books, having more space. All the more than fifteen hundred pages and illustrations are prolific and to the point. The sections devoted to bone tuberculosis are unusually instructive, while those devoted to the various forms of arthitis, acute and chronic, traumatic, infective, acute suppurative, typhoid, gonorrhoeal, pneumococcic, acute rheumatism, the chronic type, gouty, chronic osteo-arthritis and osteochondritis are replete with suggestions as to appropriate treatment. In many of these the importance of early radical measures is stressed as offering the best end resuts.

THE SURGICAL CLINICS OF NORTH AMERICA (Issued serially, one number every other month.) Volume V, Number III (Mayo Clinic Number—June, 1925.) 260 pages with 115 illustrations. Per clinic year (February 1925 to December 1925.) Paper, $12.00; Cloth, $16.00 net. Philadelphia and London: W. B. Saunders Company.

This issue is noteworthy as coming from one of the World's greatest clinical and operative centers, the Mayo Clinic. Among the articles which should be useful to the physician are: "Certain Difficulties Presented by Disease of the Biliary Tract", E. Starr Judd; "Lesions of the Stomach and Duodenum", Donald Balfour; "Surgical Treatment of Lesions of the Hip and Knee and Nonunion of the Radius", Melvin Henderson; "Surgical Treatment of Chronic Lesions of the Bone", Henry Meyerding; "Sacrococcygeal Cysts and Sinuses", James C. Masson; "Notes on Technical

Difficulties of Surgery of the Thyroid", John Pemberton; "Empyema", Harrington and Plankers; "Ureteral Stones", Bumpus and Scholl; "The Diagnosis and Treatment of the More Common Diseases of the Anus, Rectum, and Sigmoid". Louis A. Buie, and "Certain Diagnostic Problems in Otology", by Lillie and Anderson. The entire volume is splendidly illustrated, all originals.

THE SURGICAL CLINICS OF NORTH AMERICA (Issued serially, one number every other month.) Volume V, Number II (New York Number—April 1925.) 337 pages with 105 illustrations. Per clinic year (February 1925 to December 1925.) Paper, $12.00; Cloth, $16.00 net. Philadelphia and London: W. B. Saunders Company.

PHYSICAL DIAGNOSIS OF DISEASES OF THE CHEST. By Joseph H. Pratt, A.M., M.D., and George E. Bushnell, Ph.D., M.D. Octavo of 522 pages with 166 illustrations. Philadelphia and London: W. B. Saunders Company, 1925. Cloth, $5.00 net.

ORTHOPAEDIC SURGERY

Edited by Earl D. McBride, M. D.
1006 First Nat'l Bank Bldg., Oklahoma City

CLINICAL CASE REPORT: Spastic flat foot due to Gonorrheal Arthritis.

E. J. F. admitted to St. Anthony Hospital June 26, 1923, age 20, white, clerk in shoe store.

Chief Complaint: Painful feet.

History: In starting to obtain the history in this case the patient began to speak of his arches being broken and of what he thought was the cause. He had been told by his physician that his pain was due to broken arches. The history, however, revealed that one year previous to admission he had had an attack of acute gonorrhea and had cured himself in six days with a drug store remedy. Epididymitis followed. Pain in his ankles and feet came on gradually and he began to try various kinds of arch supports and shoes but everything that he tried made his feet much worse. He finally had to give up his position in a shoe store because it required so much standing. He was told that he had rheumatism and took some baths at the springs but it did not seem to releive his feet. The feet have become gradually stiffened in the joints and at the present time he can only walk for a very short distance without resting. There has been no evidence of gonorrhea since the first eight weeks of the disease.

Examination: This well developed young man walks as if stepping on something he is afraid of breaking. He has very clumsy arch supports in his shoes, modified and padded with cotton and adhesive. The feet are of normal size and proportion and the joints are not swollen or enlarged but the skin has a slight bluish color and return of color after pressure is very sluggish. All the joints are stiffened to the extent that practically no motion can be obtained in any of the joints below the ankle.

The longitudinal arch is flattened with the inner border of the foot bulging and the toes on both feet permanently dorsiflexed with the metatarsal arches convex instead of concave. G. U. reports, massage of prostate negative.

X-ray: There are spurs of bone on the plantar aspect of both heels and all of the points appear more or less atrophied and cloudy.

Operation: June 28, 1925. Ethylene anesthetic. Both feet were forcibly manipulated over a wedge so that all the joints had practically normal motion. Casts were applied from the toes to the knees with both feet forcibly inverted and toes flexed. On the fifth day after the operation, patient was able to stand and walk some in his casts. Three weeks after the operation the casts were removed and plaster impressions made for

SLIGHTLY MODIFIED WHITMAN BRACE MADE OF DURALUMINUM METAL, THE WEIGHT OF WHICH IS ONLY TWO OUNCES.

combination Whitman braces. The feet were then strapped with adhesive so as to maintain correction of the arches and a pad of felt about one-half inch thick placed back of the heads of the metatarsals and strapped on with adhesive. Daily manipulation of the feet was accomplished although the procedure was more or less painful. The Whitman Braces were applied to arches and proper shoes fitted, after which strapping was omitted but the manipulation was continued daily for two weeks. Patient was now given exercises such as walking on tiptoes and walking on the outer border of the feet. Also standing and flexing the toes on the floor twenty-five times. He was advised how to manipulate his own feet and keep the joints pliable.

Results: After two years the patient is now able to walk as far as he wishes, or stand for as long hours as is necessary. He still wears the braces, however, and states that at times flighty pains pass through his feet.

Discussion: It is no use attempting to put a rigid brace or arch support upon a foot of this kind until after manipulation under an anesthetic. In the earlier steps physiotherapy and massage together with strapping and padding of the feet may prevent adhesion and distortion of the arches. The focus of infection should be removed as quickly and as completely as possible. Manipulation under ether will fail unless focus is removed. The important feature of the operation is to completely free all adhesions. If the manipulation is not done in a severe forceful manner results will be very unsatisfactory. Fit of the brace is also a very important feature. It should be adjusted until there is no pressure from the edges and the position should permit correction of the foot without undue pressure. After care

of the feet by the patient is also very important. He should persevere in manipulating all of his joints each day and in the active exercises.

II. Fracture of the Spine.—Emil S. Geist, Minnesota Medicine, January, 1925.

The paper does not consider isolated fractures of the transverse processes, laminae, or spinous processes, nor pathological fractures.

Seventy-four cases are reported. Nearly all occurred in the active period of life (twenty to thirty-five years of age) and in males. One-third had paralysis, in most cases total, below the involved spinal segment; in a few, a partial paralysis of an extremity. With paralysis present the diagnosis is easy, but in those without parlysis it was missed in about twenty-five per cent of the diagnoses subsequently made by careful X-ray examination. The X-ray examination should include lateral and stereoscopic antero-posterior views. Fracture of the spine by no means always presents unmistakable signs and symptoms, occasionally giving no symptoms whatever, at a late date. The causative trauma need not be of extreme severity. Disability was greatest in the dorso-lumbar spine and ranged from 40 to 100 per cent. Forty-five per cent showed deformity, usually kyphosis. The complaints of the patients, in order of frequency, were: rigidity of the back, inability to lift heavy objects, weak back, pain, and paralysis. Of twenty-two cases seen within the first forty-eight hours, twelve were operated upon (laminectomy). Four of these were benefitted by operation, two died on the table, two shortly afterward, and four were not benefitted. The author feels that laminectomy offers no brilliant hope of relief of paralysis, but that without this operation the outlook is still more gloomy. There were thirty-eight "old" cases in the series, twenty-five per cent having been diagnosed as "back-strains". They complained of pain, rigidity, and inability to lift. Most of them were relieved by the application of braces. One had a spinal fusion, with complete relief.

For fresh cases, without paralysis, the author advocates rest in recumbency, best on a Bradford frame, for ten or twelve weeks, followed by a cast and later a brace. In late cases, in good operative risks, he advocates a fusion operation: where operation is impracticable, fixation by bracing.

III. Fractures of the Hip.—Melvin S. Henderson-Minnesota Medicine, December, 1924.

Only intra-capsular fractures are considered. In these the fractured surfaces are bathed in synovial fluid, and this the author considers to be the factor inhibiting callus formation. Moreover, the proximal fragment has no muscular or ligamentous attachments and very small blood supply, its nutrition being correspondingly scant. Frail, elderly people may sustain fracture by indirect violence, the fall following the fracture.

Impaction may make walking possible after fracture, and fracture should be excluded only after careful radiographic examination. An important sign is the inability (except in firmly impacted fractures) to lift the heel from bed or table with knee extended. The classical clinical sign of fracture may be absent if the fragments are well impacted. It is unsafe to trust to impaction to produce union. Some impacted fractures do unite, but many eventuate in non-union and absorption of the neck. The author considers the abduction method of Whitman the method of choice, the best means of securing anatomic restoration and fixation. The old belief that most fractures of the hip fail to unite must be revised. Only in patients with very little resistance, who cannot stand confinement, need this be true. Impaction should be broken up, the leg pulled down to normal length, and the hip swung out to full abduction, with foot in inversion. In this position the iliofemoral ligament is tightened and steadies the fragments. The position is maintained by a double plaster spica, or by traction in adduction, or by a Thomas splint. The Ruth-Maxwell traction method, if properly used, gives good results.

Reports of follow-up after the use of the Whitman method shows a good proportion of bony union and a very high percentage of good functional results.

TUBERCULOSIS

Edited by L. J. Moorman, M.D.
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Observations on Partial Artificial Pneumothorax in Early Pulmonary Tuberculosis.—M. J. Fine, The American Review of Tuberculosis, January 1925.

The author feels that partial artificial pneumothorax is very useful in early pulmonary tuberculosis and urges its use in all early cases which are either not doing well under routine management or which develop clinical symptoms and physicial signs on slight exertion.

The complications incident to artificial pneumothorax are lessened and the length of treatment necessary to obtain improvement greatly shortened when the treatment is used early in the disease.

Studies of the Respiratory Organs in Health and Disease. 20. The Value of the Vital-Capacity Test in Artificial Pneumothorax.—J. A. Myers and William Bailey. The American Review of Tuberculosis, January, 1925.

Artificial pneumothorax has a definite effect upon the vital capacity of the lungs; the extent of the collapse and the condition of the uncollapsed lung tissue help to determine this effect. Since there is a large margin of safety in the lungs, all factors of respiration mechanical and chemical are practically normal in persons with a collapsed lung and there is no dyspnea except after exertion demanding more than a threefold increase in the normal ventilation.

Readings vary greatly on pneumothorax cases, the vital capacity being reduced from 32% to 77% of the individual's theoretical normal in this series of cases. Extra-pleural thoracoplasty, of course, greatly reduces the vital capacity permanently.

Vital capacity readings are of great value both in controlling fillings and in determining the condition of the better lung. Much dyspnea and discomfort may be avoided in patients with a low vital capacity before treatment by introducing small amounts of air every twenty-four hours until compensation has taken place, subsequent fillings reducing the vital capacity very slightly. After the lung is well collapsed the vital capacity

should remain approximately stationary after refilling if the opposite lung is holding its own. A gradual reduction in vital capacity should make the operator suspicious of the contrateral lung.

Contralteral Exudative Pleuritis Complicating Artificial Pneumothorax. —Andrew Peters, The American Review of Tuberculosis, Jan., 1925.

Very few cases of contralateral exudative pleurisies are reported in medical literature, and most of these have been noted since 1920. Three cases are reported here which came under observation at Loomis among the 229 patients who have received artificial pneumothorax there. While the immediate effects of this complication are very alarming, it is frequently not so serious as might be thought, two of the three cases reported here having made recoveries. It indicates a new localization of the tuberculosis process, however, which if progressive, is, of course, unfavorable. Thus although the course of the contralateral pleurisy itself is often favorable, it aggravates the patient's condition and makes for a much less favorable prognosis. Treatment depends upon the development in the contralateral lung; the exudate must usually be aspired, while the artificial pneumothorax is disturbed as little as possible.

Air Embolism and Artificial Pneumothorax. H. A. Bishop, The American Review of Tuberculosis, January, 1925.

Statistics on the incidence of air embolism following artificial pneumothorax vary from 1 in 1000 to 1 in 1500 with a mortality rate of 30 to 50 per cent. Being dependent upon the alertness and diagnostic skill of the individual operator, these figures may be misleading and a special watchfulness for the signs of minor embolic process may cause a revision of these statistics.

The symptoms in the case presented here were characteristic of cerebral air embolism, following the withdrawal of the needle and arose when a slightly positive pressure was produced after a number of fillings. The pneumothorax was never satisfactory only a small pocket of air being produced and fillings always being difficult because of adhesions. Artificial pneumothorax is especially dangerous and difficult with this type of case, manometer readings are unsatisfactory and the danger of introducing the needle into the lung or transfixing a vessel in the highly vascularized pleura is great. Autopsy revealed the presence of air in the cerebral vessels in this case. This air, the only pathological evidence of cause of death, may have been aspirated into the vessels at autopsy in spite of the precautions taken to prevent it.

The direct cause of embolus is the entrance of air into a pulmonary or bronchial vessel. This air may enter the vessel through a rupture in its walls into an elveolus, may pass directly from the needle into a pulmonary vein, or may pass into a pleural vessel after the withdrawal of the needle, fibrosis preventing the transfixed vessel from contracting. Pleural adhesions are the indirect cause of this accident. Cases with extensive pleural adhesions must be approached carefully after thorough physical and X-ray study and every precaution must be taken during each filling. Air must not be introduced until a negative manometer reading with definite oscillations is obtained, the location of the needle must be definitely known and the operation must be stopt on the slightest sign of trouble. Each case must be carefully checked with the X-ray at frequent intervals.

The treatment for this accident is symptomatic and is not successful in well marked cases.

BACTERIOLOGY and PATHOLOGY

Edited by Wm. H. Bailey, A.B. M.D.
Wesley Hospital, Oklahoma City

FURTHER OBSERVATIONS ON THE SURVIVAL OF BONE AFTER REMOVAL FROM THE BODY.—S. L. Haas, M.D., San Francisco, Calif. Archives of Surgery—Vol. 10, No. 1, Part 1, Jan. 1925.

The author repeats the already known fact that molecular death of the body does not ensue immediately upon the somatic death of an individual. Some tissues remain viable longer than others. The period of survival is also dependent upon other factors and it is possible by certain methods of treatment of tissues removed to prolong its viability.

In discussing this question in its relation to bone tissue the author adopts the criterion of the formation of callus by the osteogenetic cells as proof of the life of the bone. In his experiments the bone was factured, removed from the animal, subjected to different treatments especially freezing temperatures for varying periods of time and then the two fragments placed in normal anatomical relation to each other and buried in the muscles of the back of the same dog from which they had been removed. After about five weeks the bones were removed and examined microscopically.

Dr. Haas makes the following conclusions:

"Utilizing as a criterion the fact that there was a physiologic reparative response of healing of a fractured bone buried in muscle, the following conclusions are presented as a result of this experimental work.

"1. There were definite signs of proliferation in fractured bones that were kept at a freezing temperature for as long as five days. The shorter the exposure period, the greater was the reparative response.

"2. Control bones that were fractured and kept at room temperature showed no signs of proliferation on exposure for two days or longer.

"3. Bone after removal from the body does not die immediately, but retains its vitality for various periods depending on the environment. The degenerative process can be retarded, but not completely suppressed, by exposing the bone to a freezing temperature.

"4. In order to insure the preservation of the maximum life properties of bone after removal from the body, it is advisable to keep it at a freezing temperature. It is also essential, that such bone be used at an early time, as the vitality of the osteogenetic cells become less as the time increases and is absent after five days."

The question was raised in the discussion as to whether or not the osteogenesis did not come entirely from the new blood vessels pushing into the bone tissue from the outsider rather than from the bone graft itself.

THE SIGNIFICANCE OF UROBILOGEN IN THE URINE AS A TEST FOR LIVER FUNCTION.—George B. Wallace, M.D., and Joseph S. Diamond, M.D., New York Archives of Internal Medicine, June, 1925, No. 6.

The test is dependent on Erlichs aldehyde reaction and consists of a series of dilutions of the urine carried to the point where no further reaction takes place. The end result is read off where the fainest pink is still discernible. Techique of the test—add lcc of urine to 20, 30, 40, 50cc of water or more. 10 cc of each dilution is placed in a test tube and to each is added 1 cc of Erlichs reagent. Read in 5 minutes, the end result is where the slightest pink color is still discernible.

Animal experiments indicate: (1) that, in parenchymatous changes of the liver, there occurs a marked increase in the urinary urobilogen; (2) that, urobilogen is formed normally in the intestinal canal by a process of decomposition of the bile pigment. Only in rare instances of intra hepatic infection, as in cholangeitis, may the urobilogen be formed within the biliary radicles.

The clinical significance of urobilogen: (1) Disease of the liver and biliary tract. (a) In catarrhal jaundice, there is an excessive amount of urobilogen formed, more at the beginning of the disease than toward the end. (b) In obstructive jaundice the urobilogen never appears in the urine. (c) Septic Cholangeitis causes an excessive amount of urobilogen to be excreted. (d) In chronic liver affection, one does not find urobilogen as a constant factor. (e) In cholecystitis and cholelithiasis, there is no alteration in liver function in the majority of cases. (2) Hemolytic disease: (a) In pernicious anemia, the presence of urobilogen differentiates the primary from the secondary form, there is a greater increase in the primary form. (b) the increase of urobilogen in malaria is a constant factor. This forms an important factor in the differential diagnosis between malaria and other infectious diseases.

THE SUGAR CONTENT OF THE CEREBROSPINAL FLUID AND ITS RELATION TO THE BLOOD SUGAR.—George M. Goodwin, M.D., and Harold J. Shelley, M.D., New York, Archives of Internal Medicine, Feb, 15, 1925, No. 2.

The sugar content of the cerebrospinal fluid is not constant in the same individual.

There is no level of sugar content of the cerebrospinal fluid which, within reasonable limits, might be considered normal. In the ordinary cases the figures varied from 40-77 mg. per hundred cubic centimeters of cerebrospinal fluid. In disturbances of the central nervous system the sugar content varied from 47-105 mg. per hundred cubic centimeters.

There is a definite relation between the cerebrospinal sugar and blood sugar. The percental relationship between the two, is relatively constant, lying between 45 and 65 per cent.

The ingestion of carbohydrates in sufficient quantities causes a definite and constant rise in the sugar content of the cerebrospinal fluid and disturbs the usual relationship between the sugar content of the cerebrospinal fluid and that of the blood.

Meningitis, whether tuberculous, staphylococcus or meningococcus frequently gives a low sugar content of the cerebrospinal fluid both as to actual milligrams per hundred cubic centimeters and in relation to blood.

The literature shows a high sugar content of the cerebrospinal fluid in relation to the sugar content of the blood in cases of encephalitis.

Untreated cases of early cerebrospinal syphilis give a low actual and relative sugar content of the cerebrospinal fluid. Under treatment this figure returns to normal.

The new plant of the Abbott Laboratories, pictured below, and now nearly ready, will be, when occupied, the finest complete pharmaceutical and reseach plant in the world. Here the newest synthetic, medicinal chemicals are made in large quaniities by improved processes, insuring purity

and accuracy. Here also are extracted from the crude drugs the medicinal principles used largely throughout the pharmaceutical industry as well as by the medical profession.

Larger quarters will be provided for the extensive research work now being carried on by a large staff of chemists and new buildings are being provided for the manufacture of the well-known Abbott pharmaceutical specialties.

The administrative office of The Abbott-Laboratories, located for many years in Ravenswood, will be moved about October 1st of this year to the new plant. The postoffice address will be Waukengan Ills., 25 miles north of Chicago, on the C. & N. W. R. R. About 24 acres of ground are owned by the Abbott Company to provide for the future expansion of their business.

"RHEUMATISM" PAINS TRACED TO FLAT FEET

Many persons think they have rheumatism of the legs or back when they have flat feet or broken arches, according to Dr. Solomon Strouse in the May *Hygeia*.

A young man fell as he was escorting his lady down the aisle of a theatre. Much embarrassed and fearing hims the victim of serious rheumatism, he stayed in bed nursing his illness. When he finally called a physician, his flat feet were discovered and his illness disappeared in a pair of properly adjusted shoes.

OFFICERS OKLAHOMA STATE MEDICAL ASSOCIATION

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Meeting Place, Oklahoma City, May 1926.
Delegates to the A. M. A. Dr. Albert Cook, Palace Bldg., Tulsa, 1925-26; Dr. McLain Rogers, Clinton, 1926-27.

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Meetings held on second Tuesday and Wednesday in January, April, July and October, Oklahoma City. Do not address communications concerning State Board examinations, reciprocity, etc., to the Journal or to Dr. C. A. Thompson, Secretary, but to Dr. J. M. Byrum, Shawnee, Secretary of the Board.

The applicant for license, either by examination or reciprocity shall be a graduate of a medical school, the requirements of which for graduation shall have been, at the time of graduation, in no particular less than those prescribed by the Association of American Medical Colleges for that particular year.

Reciprocal relations have been established with Missouri, Colorado, New Jersey, California and Louisiana, on basis of examination only, Arkansas, Georgia, Indiana, Iowa, Kansas, Kentucky, Michigan, Mississippi, Nebraska, Nevada, New Mexico, North Carolina, Ohio, Tennessee, Texas, Vermont, Virginia, Washington, Wisconsin, West Virginia, on basis of a diploma and a license without examination in case the diploma and the license were issued prior to June 12, 1908.

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It Is a Family Duty to Carry a Medical Protective Contract

The necessity is emphasized by the facts in file No. 03596. The following was received from our local attorneys, while the case was in process of litigation:

> "I beg to advise that today Mrs. ———, the wife of your assured in this case, called me by phone and advised that her husband, Dr. ———, had died on March 24th.
>
> "The case is now pending on demurrer and it is not likely that much if anything will ever be done with it, although of course they can go ahead and have the administrator or executor substituted."

After a lapse of six months the widow was served with a summons and in advising us, said among other things:

> "The Medical Protective Co.,
> Fort Wayne, Ind.
> Gentlemen:
> .....
> I suppose they think that my husband, Dr. ———, left a lot of money. The whole thing does not amount to Five Thousand Dollars, and I have three small children to raise."

The Doctor dead and the defense handicapped because he is not present to prove the propriety of his treatment; the widow financially unable to pay for own defense and endure a judgment; the raising and educating of three children dependent upon the wisdom of the doctor in carrying a Medical Protective Contract.

Twenty Percent of Wisdom Consists of Being Wise in Time.

For Medical Protective Service have a Medical Protective Contract

THE MEDICAL PROTECTIVE COMPANY OF FORT WAYNE, INDIANA